Expert Report Regarding Status, Etiology, and Causes of Mink Losses in the Jonsson Claims

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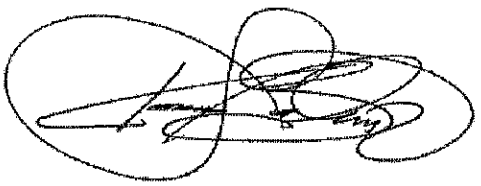

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1.0 Introduction

This report provides information and data that are relevant and in relation to the claims suggesting a potential feed issue causing adverse issues in mink. Claims have been made that Lactation Crumlet feed supplied by National Feeds, Inc (NFI) was of poor quality, causing significantly increased mortality in mink owned by Keith Jonsson and Michael Jonsson. The following sections of this report provide responses to claims proposed by the Jonsson representation including reports by Dr. Hall and Dr. H. Hildebrandt.

In the letter from Kesler and Rust (representation for Mr. Jonsson, mink rancher) dated 26 January, 2011, it states that mink feed purchased from National Feeds Inc (NFI) was rancid. It is this feed that is in question and claimed to be the causative etiology for excessive mink kit loss observed on the Keith and Michael Jonsson farms during the breeding/production phase of 2010. It is known that lactation crumlets (purchased from NFI which was produced at the Rangen plant in Buhl, ID) were added at a rate of 20 per cent into a wet-feed diet for ranch mink obtained from the Fur Breeders Agricultural (Fba) Co-op.

Relevant and appropriate steps have been undertaken to address these claims. These steps have included proper identification of potential issues and assessment of any potential risks due to claims that NFI mink feed (specifically lactation crumlets) was rancid. Valid and appropriate scientific evaluations were conducted to investigate these claims and determine if reported impacts could be substantiated by credible scientific evidence.

Extensive efforts have been made to determine not only the causative etiology of the losses reported in mink on the Jonsson farm, but also to evaluate the contributing factors that may or may not compound the issues reported in this situation. As there are numerous reasons/factors that can physiologically contribute to the numbers of losses reported in mink farming, laboratory reports, necropsy reports, and other evidence was evaluated to determine the contribution of potential factors. These links/factors have been carefully examined and weighted.

This report provides an evaluation of the available data, investigations, scientific literature, and laboratory evidence to determine the overlying cause of mortality reported from the Jonsson farms. The following bullet points provide a summary of the report:

- The review of claims of significant mink loss and the contributing etiology based on necropsy reports was determined to be substantially due to viral infections;

- Vitamin E deficiency as the primary contributing factor of the mortality on the Jonsson farms was dismissed, as evidence did not support such a claim. Evidence could not support that there was dietary deficiency of vitamin E based on the feed or the mink evaluations;

- The claim of feed rancidity in this particular case was not substantiated and was not supported or validated with reliable scientific evidence;

Therefore, it is the conclusion of this report based on the multiple factors that provide weighted evidence of the etiology of mink losses reported by Jonsson that mink losses were not the result of low quality feed, vitamin E deficiency, histamines, ionophore toxicity, or nitrosamine exposure. The following sections of this report outline the review of information and the determinations of available information to base these conclusions.

2.0 Claims of Mink Loss

Based on the cumulative findings in the summarized necropsy reports from UVDL, the primary cause of death identified in the majority of the mink was Aleutian Disease Virus (ADV) and conditions associated with the ADV disease process. Other significant findings included mink enteritis virus (MEV), hemorrhagic pneumonia, and bacterial infections that were related to pulmonary conditions. No signs of vitamin E deficiency were identified in the necropsy reports available.

2.1 IDENTIFIED CAUSES OF MINK LOSSES

Diagnostic determinations for mink were confirmed by the analyses and evaluations of several mink that reportedly died from ADV. In additional to the ADV and MEV diagnoses, other conditions reported in Jonsson mink submitted to UVDL also included the following observations:

- Three out of three mink were diagnosed with hemorrhagic pneumonia;
- Five black mink kits submitted for necropsied, all had hemorrhagic pneumonia and all were positive for ADV;
- 60-80 mink were reported to have with milky eyes, of which Dr. Larsen tested 10 mink, and all were positive for ADV using the stick test;
- Submitted mink were all positive for viral enteritis; and

- Out of four mink submitted, two reported positive for ADV, and two were diagnosed with severe gastroenteritis.

Additionally, there were no diagnoses or suggestions of vitamin E deficiency that were provided in the necropsy reports, such as myodenerative processes in muscle tissue. As such, vitamin E deficiency was not confirmed in any of the mink submitted for evaluation, or in feed samples that were analyzed for in this case. At this point, the evidence does not support the claim that mink died of vitamin E deficiency caused by poor quality or rancid crumlet feed. Not one of the mink necropsied had the reported cause of death determined as vitamin E-related.

As described, vitamin E concentrations less than (<) 2 parts per million (ppm or milligrams per kilogram [mg/kg]) suggest a chronic condition of vitamin E deficiency. If chronic vitamin E deficiency was the case with the particular mink reported herein, the body weight and condition would present as poor condition and suspect more anorexic (being off feed for an extended period of time), as well as gross pathologic conditions being reported. As this was not the case as observed in mink from the Jonsson Ranch. The overall body condition suggests that mink were receiving adequate levels of nutrients required for proper health, including vitamin E. As only 20 per cent of feed was added crumlets to the Fba Co-op wet complete feed, one would expect that significant deficiencies would not occur as mink would still be receiving 80 per cent of the recommended daily dose, if the claims were accurate.

The claim that high histamine levels were reported for Rangen fish meal could not be substantiated. The claim that ionophore toxicity caused mass mortality at this mink ranch was ruled out, as no evidence supported such a claim. The claim that nitrosamines were present and identified at a level to cause adverse effect and thus a causative agent in the reported mink losses was also ruled out by necropsy reports and the absence of pathologies consistent with nitrosamine exposure. Thus, histamine, ionophore toxicity, and nitrosamine exposure were all ruled out based on the absence of associated effects in necropsy reports and/or feed laboratory reports.

Not only was ADV and MEV diagnosed by the laboratory for Jonsson mink submitted to UVDL, but it is known that ADV does exist on these particular farms. The specific strain of ADV on the Jonnson farms, however, is not known. Based on the laboratory evidence presented in this case, viral infections cannot be ruled out as the primary cause of the reported significant losses observed at the Jonsson Mink Farm.

3.0 Aleutians Disease Virus

Both expert reports provided by the plaintiff, by Dr. Hildebrandt and Dr. Hall, provided statements verifying the presence of Aleutian Disease Virus (ADV) at the Jonsson farms. It has been reported that abnormally high losses of mink occurred in 2010 at that ranch, and ADV cannot be ruled out as the primary cause of mortality reported as the bases of this claim. As ADV was the primary cause of death for the majority of mink that were necropsied for the Jonsson farms, it is possible that ADV caused mink losses to the extent that were described in 2010 at the Jonsson farms. Therefore, ADV has not been eliminated as the primary cause of mink losses claimed at the Jonsson farms, and thus, it cannot be concluded that the losses were due to NFI feed.

3.1 ALEUTIAN DISEASE VIRUS IN MINK

It is common knowledge that ADV causes mass morbidity and mortality in a ranch population that has no prior history of exposure. As stated above, ADV is a highly contagious viral disease that has been reported on numerous mink ranches across the United States (US), with a higher prevalence in the western US. As ADV causes severely suppressed immunologic responsiveness in mink, they can become highly susceptible to the introduction of diseases. It has been reported that through appropriate and diligent management strategies, ranches can continue to produce animals while maintaining average losses. However, if a 'new' or different viral strain of ADV is introduced to a herd of mink then significant morbidity and mortality would certainly be expected. Mink not previously exposed to ADV may be highly susceptible, such as kits. Two scenarios of ADV-associated mink loss can occur:

1. If new, naive mink are introduced into a herd with prior exposure to ADV, it would be likely that the new mink would experience a large outbreak could result in significant losses in the new mink.

2. If new mink carrying a new or different ADV strain are brought into an ADV-positive herd with an existing ADV strain, significant losses of mink would certainly occur in the herd.

3.1.1 Current State of Aleutian Disease in the Mink Farming Industry

Aleutian Disease Virus remains of great concern to the mink industry, although initially described about 56 years ago and heavily researched since that time. ADV is a significant threat to mink farms, and it appears to be more prevalent in the western US than it is in the

Eastern portions of the country. ADV research remains a priority in the scientific community and continues to be a current focus of research efforts in the mink farming industry.

Summarized from an article in the 2000 Blue Book of Fur Farming by Dr. Durrant, *Aleutian Disease: Current thought on Eradication*:

> *In 1993, Utah mink farmers began to experience a high incidence of Aleutian Disease Virus (ADV). The dark mink were the mink which seemed to be the most severely affected. Historically, dark mink that were infected with ADV suffered a more chronic, progressive form of the disease. The newly discovered cases involving; dark mink exhibited signs such as immune complex disease, pulmonary vasculitis and hemorrhage, and acute death. These signs had been previously associated only with the very susceptible Aleutian type (blue) mink. ADV associated-kit pneumonia was reported in dark herds in several parts of Utah. Dark mink were so acutely affected many of the commonly associated lesions were not observed on necropsy. Diagnosis was made via post mortem blood samples submitted for testing by counterelectrophoresis (CEP) testing or tissue examination (histopathology). These observations and clinical signs raised many questions about the type or strain of ADV affecting many mink in Utah.*
>
> *There are many identified strains of ADV. Basically, the types can be divided into two classes. Type I viruses usually result in chronic, progressive disease. Type 2 viruses result in more severe illness and acute death. Research conducted at Rocky Mountain Laboratory in Montana under the direction of Dr. Marshall Bloom resulted in isolation of yet another strain of ADV. The new isolate more closely resembles the old type I virus but has characteristics of type 2 virus. Research was conducted into the possibility of transmission of the new ADV to and from wildlife. Raccoons and skunks are considered hosts and possible vectors for the transmission of any ADV (1).*
>
> *Current industry standards for many years have used CEP testing as the preferred method to identify animals that should be culled from infected mink herds. Before the advent of CEP, ranchers used the Iodine Agglutination Test (IAT). This test detected high levels of gamma globulins in collected serums. The CEP test is considered more sensitive and more specific than IAT. But the IAT is very effective and specific when used in blue mink but has proved to be less specific when used in other mink color phases. IAT would react and identify mink as positive when mink suffer from other conditions such as enteritis, urinary infections, and abscesses.*

3.1.2 Strains of Aleutians Disease

There are a number of ADV strains that have been identified and subsequently investigated. A number of different isolates of ADV have been compared electrophoretically and serologically

(Aasted *et al.*, 1984). The Utah-1 strain that Dr. Durrant also reported on (see excerpt above), was also described by Aasted *et al.* (1984) as being a highly virulent strain.

In Fur Animal Research Newsletter (December 2003, Vol. 11, No. 4), Dr. Newman stated that ADV is:

> *"...a disease that is characterized as a chronic, progressive, non-treatable, immune-mediated disease caused by a parvovirus that particularly affects the kidneys, blood vessels, brain, eyes, and lungs. High mortality follows immune failure and terminal kidney failure."*

Fur Animal Research Newsletter has maintained Aleutians Disease topics as a research priority. In fact, the February 2011 Fur Animal Research Newsletter contained three (3) out of five (5) articles focusing on ADV and the need to continue efforts to understand the disease (progression, transmission, strains/types, etc.), in an effort to control mink losses.

3.1.3 Other Effects of Viral Infections

It has been reported that tissue levels of Vitamin E are reduced by immune dysfunction related to viral infections (Odeleye and Watson, 1991). In a murine study conducted by Odeleye and Watson (1991), they report that the loss of vitamin E is due to murine virus-induced immune dysfunction (Wang *et al.*, 1994a as reported in Liang *et al.*, 1996), which results in increased production of free radicals and lipid peroxides that are immunosuppressive and can accelerate the development of viral induced diseases. Zhang *et al.* (1999) reported that superoxide radicals including lipid peroxides are produced in greater quantities during viral infections which exacerbate nutritional deficiencies and suppress immune cell function.

Although mink studies have not knowingly been conducted to determine the species-specific responses and effects of viral infections, other species have been studied in relation to the adverse, multi-organ system effects that occur with viral infections, including nutritional deficiencies. In studies by Zhang *et al.* (1999) and Liang *et al.* (1996), vitamin E deficiency and immunosuppression were identified as significant outcomes of viral disease processes.

In the study by Liang *et al.* (1996) conditions such as anorexia, weight loss, and disease associated infections frequently progress to multiple nutritional deficiencies which tend to accelerate immunosuppression. It was also reported by Liang *et al.* (1996) that superoxide radicals, including hydrogen peroxides, hydroxyl radicals and lipid peroxides are produced at high levels when immune defenses are breached with increased exposure to bacterial mitogens

and endotoxins. These are highly reactive oxygen-containing molecules that may facilitate disease progression.

4.0 Nutritional Needs for Mink

As there are specific physiological requirements of the mink during various growth and life stages, NFI produces feeds based on appropriate formulations to meet those necessary dietary requirements and nutrition needs. The National Research Council (NRC) has published nutritional requirements for four (4) primary life stages of the mink (NRC, 1982).

Each feed type produced by NFI has specific ingredient and content guarantees, which are published on each Tag Specifications label, essentially the feed quality label that is attached to each bag of feed. The values provided on the Tags, or feed labels, are required values and/or concentrations of feed components that state that "*at a minimum*" those levels of ingredients are present within the feed. NFI follows Best Industry Standards in their formulations of mink feeds, and reportedly do add in excess of the daily requirements of vitamin E to meet such physiologic demands of mink.

4.1 VITAMIN E – TOCOPHEROL

Vitamin E is a fat-soluble vitamin that is considered to be an essential nutrient, as the dietary intake of vitamin E is essential to physiologic function in most species. The class of vitamin E is comprised of eight naturally occurring fat-soluble nutrients consisting of four tocopherols and four tocotrienols. All forms feature a chromanol ring, with a hydroxyl group that can donate a hydrogen atom to reduce free radicals and a hydrophobic side chain which allows for penetration into biological membranes. Both the tocopherols and tocotrienols occur in *alpha*, *beta*, *gamma* and *delta* forms, as determined by the number and position of methyl groups on the chromanol ring (Drevon, 1991). Of the eight forms, *alpha*-tocopherol is considered to have the highest biological activity, is the form which is most abundant in foods, and has the highest molar concentration of lipid soluble antioxidant (Drevon, 1991).

4.1.1 Functions of Vitamin E in the Body

Vitamin E is a fat-soluble nutrient that acts as both a vitamin and an antioxidant and is one of body's prime defenders against oxidation stress. It protects the lipids and other vulnerable components of the body cells and their membranes from oxidation.

Vitamin E is particularly effective in preventing the oxidation of PUFA, other lipids, as well as related compounds such as vitamin A. Thus, vitamin E protects the lipids of the membranes from oxidative damage due to highly reactive oxygen species and other free radicals. Vitamin E reduces the harmful free radicals to harmless metabolites in a process called 'free radical scavenging'.

As previously stated, vitamin E also protects fats and oils in feedstuff from oxidation and rancidity, thereby acting as a preservative.

4.2 VITAMIN E REQUIREMENTS IN MINK

Dietary requirements of vitamin E for mink as reported by Puls (1994) are 30 mg/kg, which has been reported to be adequate for normal growth. In NFI mink feeds, 60 to 80 mg/kg vitamin E is normally added to the diet. This higher vitamin E level is advisable since fat ingredients, such as fish or poultry that are commonly used in mink feeds are high in PUFA.

Mink requirements of *alpha*-tocopherol were initially reported by Stowe and Whitehair (1963) as approximately 25 mg/kg of diet; this is equivalent to 0.66 mg/100 kcal of metabolic energy (ME) (NRC, 1982). It is important to note that when mink diets contain rancid fats or are high in PUFAs, the animals are subject to a disorder identified as yellow fat disease[1]. However, with an understanding of daily requirements of vitamin E, this disease manifestation is not as common as it used to be 20 to 30 years ago. As such, mink receiving such diets require an adequate supply of vitamin E, especially during the growth period (Mason and Hartsough, 1951).

4.3 VITAMIN E DEFICIENCY

Numerous studies have reported on the signs of vitamin E deficiency in mink. It has been reported that vitamin E deficiency may cause neurological dysfunction, myopathies and diminished erythrocyte life span. The erythrocyte membrane tends to be susceptible to damage by lipid peroxides, or free radicals. Additionally, signs of blood in the urine can occur as a result of rancid feed consumption and vitamin E deficiency in mink (NRC, 1982).

[1] Yellow fats disease is also known as *steatitis* (Gorham and Hartsough, 2007; Hartsough and Gorham, 1949).

R. Puls (1994) in "Vitamin Levels in Animal Health" stated the following:

> *Diets low in alpha-tocopherol and high in PUFA give clinico-pathological alterations more typical of chronic steatosis than of nutritional myodegeneration (e.g., White Muscle Disease).*

Nutritional myodegeneration (also called nutritional myopathy, nutritional muscular dystrophy, or white muscle disease) can occur in animals suffering from vitamin E deficiency. Skeletal muscle in effected animals shows characteristic whitish-yellow lesions. Damage to skeletal muscles in vitamin E-deficient animals may be mediated by increased free radical activity (Phoenix *et al.*, 1990). In addition to nutritional inadequacies of vitamin E, low tissue vitamin E levels can also be caused by or triggered by such factors as stress, rapid growth, and various diseases (mink enteritis, parvovirus, etc.) that have led to increased oxidative stress (Liang *et al.*, 1996).

As stated previously, there was no evidence as stated by diagnostic determinations related to the Jonsson mink that was suggestive of even the potential of vitamin E deficiency.

4.4 DURATION OF SUSPECTED VITAMIN E DEFICIENCY DAMAGE

Because it was determined that the mink from the Jonsson farms did not suffer from adverse effects due to feed quality, but moreso from ADV and other infectious diseases, it is assumed that no long term effects are expected in these mink. To summarize the causes of death as stated earlier in this report, diagnoses stated on available necropsy reports determining the cause of death included the following three primary causes:

- ADV;
- MEV; and
- Pulmonary disease.

However, as identified in the literature, no permanent or long-term damage due to acute, limited intake of vitamin E via the diet has been observed or reported in the scientific literature. No scientific studies have reported permanent damage to tissues or reproductive performance in animals following acute vitamin E deficiency, especially with acute mild degeneration. Once adequate levels of vitamin E are reached via diet or supplementation, the adverse effects are resolved and tend to no longer be an issue. Long-term damage due to vitamin E deficiency has been reported in the literature as being dependent on factors including the duration of exposure to significantly deficient diets and the cessation.

4.5 ALTERNATIVE CAUSES OF NUTRITIONAL DEFICIENCIES

Mink farms often have non-etiologic causes of death that regularly occur in mink. Often these events can be triggered by the presence of opportunistic pathogens, including bacteria and/or viruses. These pathogens can potentially cause disease if they gain entry into the body through skin or mucosal surfaces that are damaged, if the immune system of the animal is somehow compromised, or if a sudden imbalance in the composition of the normal flora develops (Gorham, 1998). These situations can arise on mink farms if there are any management conditions that increase stress, compromise optimal nutrition, or involve the improper use of antibiotics (Gorham, 1998), or not vaccinating mink appropriately.

Various studies have clarified the relationship between loss of vitamin E, increased lipid peroxidation and immune dysfunction often caused by viral infections (*e.g.*, murine retrovirus infection). Studies have shown that Vitamin E may be an important immune modulator because tissue levels of vitamin E are reduced by immune dysfunction during disease processes (*e.g.*, murine retrovirus) (Wang *et al.*, 1994a,b,c as reported in Liang *et al.*, 1996). Further studies have demonstrated that the loss of vitamin E may be due to retrovirus-induced immune dysfunction (Wang *et al.*, 1994c as reported in Liang *et al.*, 1996), which results in increased production of free radicals and lipid peroxides that are immunosuppressive and could accelerate development of certain diseases (Odeleye and Watson, 1991).

Superoxide radicals including hydrogen peroxides, hydroxyl radicals and lipid peroxides are produced at high levels when immune defenses are breached with increased exposure to bacterial mitogens and endotoxins. These highly reactive oxygen-containing molecules may present as tissue damage caused by oxidative stress, as well as facilitate disease progression from viral infection (Baruchel and Xainberg, 1992 as reported in Liang *et al.,* 1996) by their reaction with antioxidant vitamins (*i.e.,* vitamin E). Despite feed providing the adequate recommended level of vitamin E in the diet, this process can exacerbate nutritional deficiency, as well as directly inducing immunosuppression (Liang *et al.*, 1996).

Again, there is no threat of damage in the Jonsson mink herd, as vitamin E deficiency was not identified in any of the necropsied mink, there were no nutritional degenerative lesions reported, and no feed issues were reported.

No other nutritional issues were identified in the Jonsson mink or in the feed supplied by NFI.

5.0 Mink Feed Quality

Several evaluations have been conducted to determine quality of feed, and association with reported claims of mink losses at the Jonsson farms. Investigations appropriate to determining feed quality were identified was a primary area of interest. As such, feed samples were analyzed to determine their quality.

A feed sample (ID #740-2010-00009273) from the batch feed (Batch#EUCAPE-00014440) purchased by Jonsson Mink Farm (RE: lactation crumlets 11/09/10, powder) was analyzed under the Industry Best Practices for quality assurance and quality control (QA/QC) measures. Analyses requested for determination of feed quality included the following:

- QD252: Protein – Combustion;
- QD250: Ash;
- QD226: Calories, calculated;
- Carbohydrates, calculated;
- Aflatoxin,
- Zearalenone;
- Fat by acid hydrolysis;
- Moisture by vacuum oven;
- Peroxide value; and
- Salmonella by PCR.

5.1 LACTATION CRUMLETS

The Certificate of Analysis (AR-10-KK-009658-01) provided results that clearly demonstrate the quality of the feed in that it did meet the label standards (as reported on the landscape labels on each bag of feed, the "Feed Tag"). Additionally, the quality of feed based on rancidity as measured by peroxide value (PV), was well within the range of acceptable quality with no significant deterioration of feed stuff quality determined. The Industry Standards for acceptable and safe range for peroxide formation is 1 to 10 mEq/kg[2] fat. Test results show that this feed sample was well within those limits with a reported PV of <10 mEq/kg fat. Additional parameters analyzed were also well within appropriate and acceptable limits.

[2] mEq/kg =the unit of measure is milliequivalents per kilogram of fat as measured in a feed sample.

An independent analysis of a feed sample (laboratory ID# 9664683) taken from the same batch was submitted by Mr. Kent Griffeth on 22 November, 2010. The results also demonstrate that the feed was not rancid (results reported on 30 November, 2010). The results of the PV test were reported to be 2.58 mEq/kg fat. These results demonstrate that the feed was not rancid at the time it was analyzed[3].

It is therefore summarized in concluded that the lactation crumlet, feed as a small percentage of crumlets added to the wet co-op diet, was not the cause of the conditions observed in mink from Jonsson farm.

5.2 CRUMLETS ADDED TO COMPLETE WET FEED

As part of the Utah Fba co-op for mink ranches, a complete wet feed diet is produced and available for co-op members. The Jonsson farms as well as other ranchers are known to sometimes supplement the wet feed by adding ingredients that they believe will provide their mink with additional nutrients as needed. In the case of Jonsson, NFI lactation crumlets were added at a relative rate of 20 per cent of the feed. As the crumlets are also produced as complete feeds, no deficiencies are expected from either the wet feed (co-op) or the crumlets (NFI). Additionally, hypothetically, if one of these complete feed components was reported with lower than expected levels of an ingredient, the remaining portion of diet should and would support the general needs for health and maintenance of the mink. If in fact the crumlets were tested to show lower than normal levels of vitamin E, the co-op wet feed would provide approximately 75 to 80 per cent of the recommended daily dose. This could result in a liver concentration that would be expected to be within the normal ranges, but not excessively elevated.

Feed samples are regularly submitted for analyses for QA/QC measures by the manufacturer. The analytical reports from the feed analyses are used to demonstrate that the quality of the feed meets the label standards (as reported on the landscape labels on each bag of feed, the "Feed Tag" of "Tag Specs") at the time of production and expected use of that feed, and samples are not rancid or of diminished quality.

The quality of feeds based on rancidity as measured by PV analysis should be *within the range of acceptable quality*. The Industry Standards for an acceptable and safe range for peroxide formation is 1 to 10 meq/kg fat (milliequivalents per kilogram of fat). Another measure of feed

[3] It is noted that at the time of the analysis to determine oxidation potential of feed as measured by peroxide value, the feed was in fact reported to be of high quality. Rancidity is a progressive process; the oxidation potential increases as time passes. Therefore, it cannot be claimed that feed was more rancid at an earlier date than the tests conducted at a later date.

quality is the Active Oxygen Method (AOM). The AOM parameter determines stability of the feed product, and this value should also be within the established acceptable limits, with reporting values of 20 meq/kg or less. The date of the analysis is important, as it relates to the period of time in which a feed designated as fresh is intended to be used or fed out, and thus is also related to the shelf-life of the product. As the shelf-life of a particular feed is used up and is near its expiration, this can result in an increasing PV value to as the feed gets older.

5.3 MEASURES OF RANCIDITY

This section is presented to provide background of information and as a component to understanding the feed quality issue, such as peroxide values.

Oils and fats[4] as components of feed can become rancid when they undergo a degradation process known as oxidation. Rancidity in food and feedstuff can result from oxidation, or decomposition, of the lipid component of the feed, microbiological deterioration of the sample, or both. A variety of chemical compounds such as peroxides, aldehydes and free fatty acids are created during the oxidation of oil/fats (Hamilton and Kirstein, 2008). There are two tests that tend to be employed to determine if the feedstuff has undergone oxidation, or to monitor or predict oxidative degradation. Rancidity is usually expressed as a Peroxide Value (PV), and in some cases with an additional test that is a measure of feed (oils and fats) stability using the Active Oxidative Method (AOM).

- **Peroxide Value (PV)** is the measure of the state of rancidity of a sample. Also called Initial Peroxide Value (IPV) because it is determined on a sample as submitted. The PV of unstabilized fat can change quickly. For this test, peroxides are indirectly measured under standardized conditions. The result is called the Peroxide Value, expressed as milliequivalents of peroxide per kilogram of fat (meq/kg). Fresh non-rancid fats have a low PV - usually less than (<) 5.0 meq/kg.

- **Active Oxygen Method (AOM)** is a measure of the ability of a fat to resist oxidative rancidity during storage. An oil or fat is subjected to conditions known to accelerate degradation to help gauge the sample's resistance to oxidation. Oxygen is bubbled into a fat to cause oxidation of the fatty acids. The PV test is used to monitor oxidation after the sample is stressed under controlled conditions for a long time or until a specific PV is achieved.

[4] Oils and fats affected by rancidity are primarily unsaturated fatty acids, those containing one or more bonds, such as oleic, linoleic, and arachidonic acids, resulting in disagreeable flavors and odors.

Oxidation of the fat decreases the nutritional quality of the feed. Oxidation of unsaturated fatty acids yields ketones and free fatty acids that are usually unpalatable and potentially injurious. Thus, antioxidants such as vitamin E added to feeds are used to help stabilize the oxidative reaction.

5.3.1 Oxidation

Feeds deteriorate over time, exposure to environmental factors, increased temperatures, and exposure to air – all factors or conditions that are also related to the shelf life of a feed product. The shelf life can certainly be affected if improper storage and handling of feed occur. A brief description of process of lipid oxidation is provided below.

The process of lipid oxidation is minimized by adding antioxidants or substances, such as vitamin E, which are used to preserve food by retarding deterioration rancidity or discoloration due to oxidation (21CFR(170.3(o)(3)) by donating electrons for this reaction. There are four chemical mechanisms that can decrease the rate of oxidation:

1. Hydrogen donation by an antioxidant;
2. Electron donation by an antioxidant (vitamin E);
3. Addition of the lipid to the antioxidant; and
4. Formation of a complex between the lipid and the antioxidant.

5.3.2 Protective Role of Vitamin E in Feed

Vitamin E does have an important and protective role against free-radical-mediated oxidations. Vitamin E is present in cell membranes and forms a structural complex with polyunsaturated fatty acids (PUFA) of phospholipids (Turchetto and Pignatti, 1982). Large amounts of dietary PUFA can increase the requirement for vitamin E and can also deplete stores of vitamin E in tissues.

5.4 FEED SAMPLING

Food sampling is a process used by manufacturers such as National Feeds, Inc. to check that a food is safe and that it does not contain harmful contaminants. NFI has provided documentation of these tests that are run regularly to ensure that only high quality feeds are available to clients. In addition to protecting against harmful contaminants, feed testing also provides information on:

- Whether feed contains only permitted additives at acceptable levels;
- That feed contains the right levels of key ingredients and levels of nutrients present; and

- Feed label declarations are correct.

As one would expect the manufacturer to sample legitimate and representative feeds that are of good quality, preserved appropriately, etc., as too, should feed samples be representative of the feed and conditions being analyzed. Factors relevant in considering the representativeness of a sample include:

- The conditions the sample was stored, including exposure to moisture, temperature, and duration (or expiration);
- The homogeneity of the food from which the sample is taken;
- The relative sizes of the sample to be taken and the whole;
- The potential degree of variation of the parameter(s) in question through the whole; and
- The significance and intended use of the analytical result.

Any sample taken for analysis needs to be sufficiently representative of the food for the analytical result to be meaningful and meet requirements (*e.g.*, labeling declarations, assurance of compliance with legislative or other standards, monitoring of production quality, or for routine quality control) (Ogden *et al.*, 1998).

5.4.1 Quality Assurance and Quality Control (QA/QC) of Sampling

Analytical laboratories follow strict policies and procedures related to quality control/quality assurance (QA/QC). The goal of any Analytical Laboratory QA/QC Program is to guarantee the generation of precise and accurate analytical data. Quality assurance involves the planned and systematic actions necessary to provide confidence, precision, and reproducibility in each analytical result. The QA/QC Program has two components:

- **Quality Assurance (QA)** - the system used to verify that the entire analytical process is operating within acceptable limits; how samples and data are collected and managed, and the activities which demonstrate a quality standard has been met; and

- **Quality Control (QC)** – the mechanisms established to measure non-conforming method performance; how well samples are collected and analyzed, how well the results match defined set of standards. Quality control concerns itself with those quality practices that specifically focus on product quality and the objective of laboratory QC is to produce test data whose accuracy and reproducibility are consistent with the technology involved.

6.0 Summary and Conclusions

This report summarizes the available evidence to claims of significant losses of mink at the Jonsson farms. Extensive efforts have been made to determine not only the causative etiology of the mink losses reported, but also to evaluate the contributing factors that may or may not compound any issues observed in this case. These links/factors have been carefully examined and weighted.

It was reported that lactation crumlets were added at a rate of 20 per cent to supplement a complete wet feed diet provided by the Fba co-op, even further limiting the potential for the cause of adverse effects being claimed. Lactation crumlets added to a complete wet feed, would provide more than adequate levels of the necessary nutritional needs of the mink.

There is no evidence that supports the claims that mink feed produced by Rangen for NFI feed was of poor quality or rancid. It was determined that the feed did in fact meet the necessary and appropriate health standards for the mink industry, as well as the nutritional requirements of a complete feed diet for mink. Following rigorous testing and scientific analyses, it has been demonstrated that the crumlets were superior in quality and met and/or exceeded quality standards levels for the pet food industry and feed tag specifications for ingredient quality in this case at the time it was produced and delivered.

It was demonstrated that the feed in question did meet the standards for vitamin E concentration as recommended by the NRC (1982), and would provide adequate levels of vitamin E meeting the guidelines of daily requirements for mink under normal circumstances. Based on the data provided by various test results, the valid and appropriate scientific evidence has demonstrated that the feed was in fact high quality without detection of rancidity biomarkers (elevated peroxide value).

Scientific literature has provided information that suggests additional factors can contribute to the decrease of vitamin E levels. For example, increased stress, rapid growth, or exposure to infectious agents (bacteria or viruses) have been reported as contributing to increased oxidative stress, increased consumption of antioxidants leading to the potential for tissue damage and physiological manifestations of disease processes.

Necropsy results for mink that were submitted for laboratory examination indicate numerous pathologies including ADV, MEV, and hemorrhagic pneumonia. Therefore, it is concluded that the presence of widespread viral infections (ADV and MEV) were the primary cause of health

issues surrounding the losses reported at the Jonsson farms. As such, the apparent cause of significant losses of mink at the Jonsson farms was not attributed to feed quality or the presence of ionophorous compounds or nitrosamine in feedstuffs.

Additionally, it is unclear what mink feeding practices are used at the Jonsson farms. Therefore, comments cannot be made to address the influence of these feeding factors or practices on mink health.

It has been determined that the evaluations in response to this issue provide competent and reliable scientific evidence that was unable to substantiate the claims of rancid mink feed. In conclusion, the claims of rancidity could not be substantiated or supported through valid and reliable scientific evidence.

7.0 Limitations and Closure

This report provides a review of available data, information and reports provided by the client and/or third parties. There are no assurances regarding the accuracy and completeness of this information. All information received from the client or third parties in the preparation of this report has been assumed by Stantec to be correct. Stantec assumes no responsibility for any deficiency or inaccuracy in information received from others.

Conclusions made within this report are a professional opinion at the time of the writing of this report. This report is not a legal opinion regarding compliance with applicable laws. The opinions expressed in this report are based on preliminary information obtained to date. I reserve the right to change this report as additional information may become available.

Stantec assumes no responsibility for losses, damages, liabilities or claims, howsoever arising, from third party use of this report.

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Dr. Beckett is a Senior Environmental Scientist and Toxicologist at Stantec in the Environmental Management Group who specializes in ecological risk assessments and ecotoxicology. She designs and performs ecological characterizations and sampling programs, and evaluates ecological risks in terrestrial, marine, and aquatic environments. Dr. Beckett offers particular expertise in animal science, ecotoxicology, marine biology, wildlife biology, and zoology, with experience in both academic and private industry settings. Additionally, she is known for her good relations with clients and federal and State agencies, alike.

Dr. Beckett has conducted large-scale surveys of wildlife and coastal habitats in the US from Alaska to New England, as well as numerous provinces in Canada (from British Columbia to Ontario). Dr. Beckett has performed studies of threatened and endangered species, marine mammals, large mammals (including brown bear, moose, bighorn sheep, and mountain goats), fish and invertebrate species, as well as shorebirds and avian species. Additionally, Dr. Beckett has extensive experience with reproductive and developmental toxicology in mammalian species, and she has developed an impressive record working with various constituents within several chemical classes (organics, inorganics/metals, hydrocarbons, etc.).

Dr. Beckett has conducted research with a focus on evaluating biological effects from exposure to environmental pollutants and establishing biomarkers of this exposure. A significant portion of that work included histopathological studies of polychlorinated biphenyls (PCB) exposure in mink, including laboratory and natural field studies. Her work on PCBs has made her a recognized expert in regards to environmental PCB issues, and she has been involved in several projects concerning PCBs, such as the Housatonic River, MA. Additioanl research has focused on evaluating the toxicological effects of hydrocarbons (such as weathered crude oil) including neurological, behavioral, and reproductive effetcs in wildlife. She has designed and conducted research projects that have led to a solid record of scientific achievement in toxicology, marine science, and wildlife biology.

EDUCATION

Ph.D., Dual Degree, Animal Science and Environmental Toxicology, Michigan State University, East Lansing, Michigan, 2005

M.A., Marine Biology and Ecotoxicology, University of Alaska-Fairbanks, Fairbanks, Alaska, 2000

B.A., Zoology, University of Montana, Missoula, Montana, 1991

PROFESSIONAL ASSOCIATIONS

Member, Society for Environmental Toxicology and Chemistry (SETAC)

Member, North Atlantic Chapter of National SETAC

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PROJECT EXPERIENCE

Risk Assessments

Storage and disposal facility for waste polychlorinated biphenyls (PCBs): Screening-level Ecological Risk Assessments of PCBs (Senior Project Scientist)

Stantec conducted a screening-level ecological risk assessment and biological evaluation under USEPA guidelines for a TSCA-permitted PCB-waste storage and processing facility in Nevada. Impacts caused by exposure to PCB congeners in endangered species and potential adverse impacts to representative ecological receptors were evaluated. (2011)

Railyard Off-Site Bioaccumulation and Ecological Risk Assessments of PCBs in a Tidally-Influenced River (Senior Project Scientist)

Stantec conducted a preliminary bioaccumulation assessment and ecological risk characterization for PCB-loading into a riverine system. A field sampling program was designed in support of several objectives for the site, including bioaccumulation of PCBs in fish and potential impacts to human and ecological receptors. (2008 – current)

Bulk Fuel Storgae Facility – Preliminary Ecological Assessment (Ecotoxicologist and Senior Scientist)

The Stantec team has been conducting remediation activities at a long-term fuel facility in Alaska, and recently was requested to assess the preliminary ecological impacts as a result of historical fuel releases. In support of this assessment, flora and fauna studies were also conducted, including bald eagle nest surveys within the vicinity. (2009 – current)

Neponset River Screening-Level Ecological Risk Characterization, Massachusetts* (Project Manager)

The Bird Machine Company (BMC) property is a 165-acre property that is traversed by the Neponset River in South Walpole, Massachusetts. A Stage I Environmental Risk Characterization (ERC) was conducted at the Site to address an oil spill. The Stage I ERC identified exposure pathways, assessed if target receptors were at risk from contaminant exposure, and determined if further quantitative assessment (i.e., Stage II Environmental Risk Characterization) was required for the site. Activities to support the ERC included: a site investigation; characterization of the type, volume, nature, and source of the release; identification of potential exposure pathways, points and concentrations; and determination of background levels for the chemicals of potential concern (COPCs). The ERC served as a decision tool for making remedial decisions. (2005-2006)

Callahan Mine CERCLA Site RI/FS, Maine (Senior Project Scientist)

Stantec designed and directed a large-scale ecological characterization and extensive field studies in support of risk assessment and removal/restoration activities at the site of a former zinc mine located at tidewater. Tasks included evaluations of wildlife and ecological impacts through field surveys, biota and sediment sampling, modeling, and statistical analysis. Stantec coordinated the development of an overall ecological risk assessment for the project, and provided technical support in sampling design and data analysis. (2005-2007)

Tyrolit Site Sampling and Risk Assessment, Massachusetts (Risk Assessor)

Stantec provided assistance in developing a Stage II Ecological Risk Characterization under the Massachusetts Contingency Plan (MCP) for this 162-acre manufacturing site which contained several former landfills. Stantec tasks include conducting a baseline site characterization, receptor characterization, collection of sediment, biota (invertebrates and amphibians), and surface water samples, evaluating analytical results in the context of ecological risk, and providing an assessment of risk posed by Chemicals of Potential Concern (COPCs) including volatile organic compounds (VOCs), polynuclear aromatic hydrocarbons (PAHs), volatile petroleum hydrocarbons (VPH), extractable petroleum hydrocarbons (EPH) and metals. (2005-2007)

* *denotes projects completed with other firms*

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Assessment of the Impacts to Sediment Quality in the Man-Made Pond, Dover, New Hampshire (Project Manager and Risk Assessor)
An impact assessment was completed to evaluate potential risks posed by chemicals of concerns in sediment in a man-made pond. The assessment used background information, site data and food chain modeling to address the potential for adverse effects, as well as the need for remedial measures. (2007)

Environmental Impact Statement (EIS) and Division End Points - National Energy Board EIS Review, Burnaby, B.C. (Senior Project Scientist)
Stantec conducted a review of the Draft Environmental Impact Statement (EIS) and Division End Points prepared for the fuel delivery line release, Burnaby, BC (Property). The project objectives were to: review the documents provided by the National Energy Board (NEB) and provide the NEB with a summary; determine if the EIS and Division End Points documents were consistent with Industry Best Practices; identify and document deficiencies, issues, comments or recommendations; and, provide pertinent and applicable references. This document and the information contained within, was to be used to assist the NEB in discussions in regards to a fuel delivery line release, Burnaby, BC. (2007)

Draft Environmental Impact Statement (DEIS) and Division End Points - National Energy Board Comments Summary and Review, Burnaby, B.C. (Senior Project Scientist)
Stantec provided a thorough review and summary of stakeholder comments regarding the Draft Environmental Impact Statement (EIS) and Division End Points prepared for the fuel delivery line release, Burnaby, BC. The review included: a brief summary of the submitted comments from each stakeholder; comments regarding discrepancies and consistencies amongst the submitted comments; a summary of key points from each stakeholder highlighted through the review process. The report was prepared for the exclusive use of NEB. (2008)

Neponset River Sampling and Risk Assessment: Stage II Baseline-Level Ecological Risk Characterization, Massachusetts (Risk Assessor)
As part of a Stage II Ecological Risk Characterization for this 165-acre former manufacturing site, Stantec developed work plans, performed a site characterization, and designed and conducted an extensive sampling program to evaluate risk from chemicals of potential concern (COPCs) including metals, polynuclear aromatic hydrocarbons (PAHs), and extractable petroleum hydrocarbons (EPHs). The sampling program included the collection of surface water, sediment, benthic invertebrate, and fish tissue samples. A Stage II ERA was developed looking at both terrestrial and aquatic target receptors. (2006-2007)

Neponset River Sampling and Dioxin and Furan Assessment, Massachusetts (Project Manager)
During Phase I and II investigations, dioxins and furans were detected in soil samples on site, and the Neponset River was identified as a potential transport pathway for the dioxins and furans. Statistical design and analysis was conducted to assess the nature and extent, as well as fingerprinting of dioxins/furans on and off site. The overall objective of this statistical approach was to evaluate and compare the specific chemical components measured in sediment sample to determine if the chemical, or percent similarity of congeners, was similar within all sampling locations both on and off property. (2007-2008)

Ecusta Paper Mill RI/FS, North Carolina (Project Manager and Risk Assessor)
Stantec is conducting a large scale Remedial Investigation and Feasibility Study (RI/FS) for the Davidson and French Broad Rivers which were potentially impacted by operation of the former paper mill in North Carolina. Mercury and dioxins/furans have been identified as contaminants of potential concern (COPC). Stantec is conducting biological surveys and bioaccumulation assessments, which will be incorporated into an Ecological Risk Assessment (ERA) with the support of the following studies: 1) Evaluate river stability and sediment supply within the River Investigation Area; 2) Evaluate the current

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bioaccumulation potential of mercury and dioxin/furan within the River Investigation Area; 3) Characterize sediment containing COPCs that could become bioavailable; 4) Estimate the potential for future mercury and dioxin/furan loading due to sediment disturbance within the River Investigation Area (based on fate and transport of increased sediment disturbances); and 5) Estimate the future potential for bioaccumulation of mercury and dioxin/furan within the River Investigation Area. Stantec will coordinate and provide technical support in sampling design and data analysis for the project, and develop the ecological risk assessment and support the human health risk assessment. (2008)

Stage I Environmental Risk Characterization for PCBs, Massachusetts (Senior Project Scientist)
During Phase I and II site investigations, buried drums were identified on property that is located along a brook. Chemical analysis was conducted to determine the contents of the drums; PCBs and other COPCs were detected in soil samples. A Stage I Environmental Risk Characterization (ERC) was performed that identified complete exposure pathways for the COPCs resulting in exposure to ecological receptors. The results demonstrate that a Stage II ERC will be necessary to evaluate the ecological risk to target receptors. The workplan and sampling design have been submitted for the Stage II work that will be necessary to complete on this site. (2010)

Environmental Toxicological Review for Synthetic Turf Products (Senior Project Scientist and Toxicologist)
Stantec conducted a toxicological review and summary of existing scientific literature for potential impacts of synthetic turf products to both human and environmental receptors. (2009 – 2011)

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Beckett, K.J., R.J. Aulerich, L.K. Duffy, J.S. Patterson, and S.J. Bursian. 2002. Effects of dietary exposure to environmentally relevant concentrations of weathered Prudhoe Bay crude oil in ranch-raised mink (*Mustela vison*). *Bulletin of Environmental Contamination and Toxicology, 69:593-600.*

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Beckett, K.J. et al. 2005. A unique Jaw Lesion Induced by PCB 126 in Mink. *ANS 417 (Spring) Lecture Series, Topics in Toxicology; Michigan State University*, 2005.

Beckett, K.J. et al. 2004. Mink as an Animal Model of PCB 126-Induced Carcinogenicity. *ANS 407 (Fall), Lecture Series, Topics in Toxicology; Michigan State University*, 2004.

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Beckett, K.J. et al. 2003. Does In utero Exposure to Crude Oil in Ranch-Raised Mink (Mustela vison) Kits Induce Neurotoxicological Consequences? *University of Alaska-Fairbanks, Basic Neuroscience Program Seminar, Invited Speaker*, 2003.

* *denotes projects completed with other firms*

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Beckett, K.J. et al. 2002. PCB 126 induces mandibular and maxillary psuedocarcinomatous hyperplasia and reproductive dysfunction in mink (Mustela vison). *Proceedings Society of Environmental Toxicology and Chemistry (SETAC) Conference (Abstract)*, 2002.

Fee schedule for kjb

Kerrie Beckett, PhD

2. Fee schedule or compensation to be paid for the testimony;

Fee Schedule: Compensation to be Paid for the Testimony		
Document Review and Preparation	$ 167.00	Hour
Deposition and Testimony	$ 200.00	Hour
Travel	Time and Expenses	NA
Mileage	Government Rate	Mile
The rates provided herein are based on Stantec Consulting Inc., rates for Employee Level and Status. These rates have been negotiated and are under contract for the duration of Services.		